THE ARTERIAL BLOOD PRESSURE

Learning objectives:

- List the factors that affect arterial blood pressure
- Describe the mechanisms by which arterial blood pressure is regulated
- Indicate the relationship between arterial pressure, cardiac output, and total peripheral resistance and Predicts how arterial pressure will be altered when cardiac output and/or total peripheral resistance changes.
- Given arterial systolic and diastolic pressures, estimates mean arterial pressure.
- Indicate the relationship between pulse pressure, stroke volume, and arterial compliance and predicts how pulse pressure will be changed by changes in stroke volume, or arterial compliance.

Arterial Blood Pressure (ABP)

- **Systolic blood pressure** is the peak pressure reached during systole in the aorta and other large arteries. It is about 120 mm Hg (normal range is 90-140 mm Hg) in adults.
- **Diastolic blood pressure** is the lowest pressure during diastole and is about 80 mm Hg (normal range is 60-90 mm Hg) in adults.
- **Mean arterial pressure** is the average pressure throughout the cardiac cycle. It approximately equals the diastolic pressure plus one-third of the pulse pressure (about 90 mm Hg in normal adults).

Mean arterial pressure = Diastolic pressure + 1/3 pulse pressure

Mean arterial pressure does not equal the arithmetic mean because systole (during which pressure rises) is shorter than diastole (during which pressure decreases), therefore the mean pressure is lower than the arithmetic mean i.e. nearer to the value of diastolic pressure. However, when heart rate is rapid the cardiac cycle becomes shorter. Shortening affects diastole more than systole. Under such conditions, mean arterial pressure becomes nearer to the true arithmetic mean.

- **Pulse pressure**: it is the difference between the systolic and diastolic pressure. It is normally about 30-50 mmHg.

Determinants of mean arterial blood pressure:

Mean arterial pressure (MAP) – Central venous pressure (CVP) =

cardiac output (CO) × total peripheral resistance (TPR)

$$MAP - CVP = CO \times TPR$$

by re-arrangement:

$$MAP = (CO \times TPR) + CVP$$

Since cardiac output = stroke volume (SV) × heart rate (HR)

Then,

$$MAP = (SV \times HR \times TPR) + CVP$$

Accordingly, the following factors determine the arterial blood pressure:

1- Stroke volume:

Big stroke volume elevates ABP. It increases the systolic more than the diastolic pressure and therefore the pulse pressure is increased.

2- Heart rate:

Increased heart rate elevates ABP. It increases the diastolic pressure more than the systolic because less time is available

for drop of pressure as the diastole is shortened. The pulse pressure is decreased.

3- **Total peripheral resistance:**

Peripheral resistance is mainly caused to resistance of the arterioles. Increased TPR elevates ABP. It increases the diastolic more than systolic pressure, therefore the pulse pressure decreases.

4- **Arterial compliance:**

If arterial compliance decreases e.g. due to atherosclerosis, systolic pressure increases because arteries are not able to distend enough to accommodate the stroke volume. Diastolic pressure decreases because the ability of the arteries to recoil in diastole is decreased. Pulse pressure therefore increases.

5- **Central venous pressure:**

Higher central venous pressure leads to elevation of MAP while lower central venous pressure has an opposite effect.

Physiologic variations in ABP:

1- **Age:** ABP generally increases with age. Normal value of ABP in infants it is about 80/40 mm Hg. In children, it is about 100/65 mm Hg. In young adults, it is about 120/80 mm Hg. In old age, it should not rise above 140/90.

2- **Sex:** Below the age of menopause, women usually have lower ABP than men of the same age. However, after menopause ABP rises in women due to hormonal changes that occur after menopause.

3- **Race:** people belonging to some races may have higher ABP and higher incidence of hypertension than people belonging to other races.

4- **Circadian rhythm and ABP:** In normal persons (working at daytime), ABP reaches peak value early in the morning and decreases to its lowest level at midnight. These variations in ABP between daytime and nighttime may amount to 15 - 25 mmHg. The nocturnal ABP changes follow sympathetic nervous system activity, which is minimal during nocturnal sleep, and increases on awakening. The opposite occurs in night-workers.

5- **Emotions:** strong emotional stress elevates ABP.

6- **Muscular exercise:** changes in ABP during exercise depend on the type of exercise. If exercise is dynamic (involving alternating contractions and relaxations) systolic pressure is moderately increased while diastolic pressure either falls or not changed. If the exercise is static (involving continuous constant muscle contraction), then both systolic and diastolic pressures are markedly increased.

7- **Effect of gravity on ABP:**

- The MAP in all major arteries is about 100 mm Hg when they are at the level of the left ventricle e.g., when the subject is lying down.

- However, in standing position pressure in arteries above the level of left ventricle decreases by 0.77 mmHg for each 1 cm above the level of the ventricle. Pressure in arteries below the level of left ventricle increases by 0.77 mmHg for each 1 cm below the level of the ventricle.

  For example:

  - The MAP in a large artery 50 cm above the heart equals:

    $$(100 - [0.77 \times 50]) = 62 \text{ mm Hg.}$$

  - The MAP in a large artery 105 cm below the heart equals:

    $$(100 + [0.77 \times 105]) = 180 \text{ mm Hg.}$$

Regulation of Arterial Blood Pressure

Regulation of arterial blood pressure is achieved by three groups of mechanisms that differ in their time course:

1- Rapidly induced mechanisms:

These are ***nervous mechanisms***. They comprise a group of *reflexes* that act rapidly (within seconds to few minutes) affecting the level of arterial blood pressure.

2- Intermediate time course mechanisms:

These mechanisms take longer time to operate (minutes to few hours). They involve

a- Capillary fluid-shift response

b- Renin-angiotensin system.

c- Hormonal

3- Long term mechanisms (RENAL) :

These mechanisms develop over several days and are important in long-term regulation of blood pressure . They include:

a- Renal pressure natriuresis.

b- Aldosterone secretion.

Nervous Regulation of Arterial Blood Pressure:

The Rapidly Induced Reflexes:

The centers of these reflexes are present in medulla oblongata of the brain stem. These medullary centers are the “vasomotor area” and the “cardiac inhibitory area” (figure 1)

I. Vasomotor area:

- This area contains neurons that mediate sympathetic discharge to blood vessels and heart.
- Neurons of this area discharge directly to sympathetic preganglionic neurons in the intermediolateral gray column (IML) of the spinal cord. These neurons discharge to sympathetic postganglionic neurons in sympathetic ganglia. Postganglionic noradrenergic neurons innervate the heart and blood vessels.
- Stimulation of vasomotor area leads to **elevation of arterial blood pressure** through the following mechanisms:
  1. Arteriolar constriction increasing total peripheral resistance TPR.
  2. Venoconstriction: this helps venous return and hence cardiac output.
  3. Increased heart rate and stroke volume thus increasing cardiac output.

4. Associated decrease in the tonic activity of the vagal fibers to the heart.

II. <u>Cardiac inhibitory area</u> .

- Neurons in this area mediate vagal discharge to the heart.
- Stimulation of this area decreases heart rate and cardiac output.

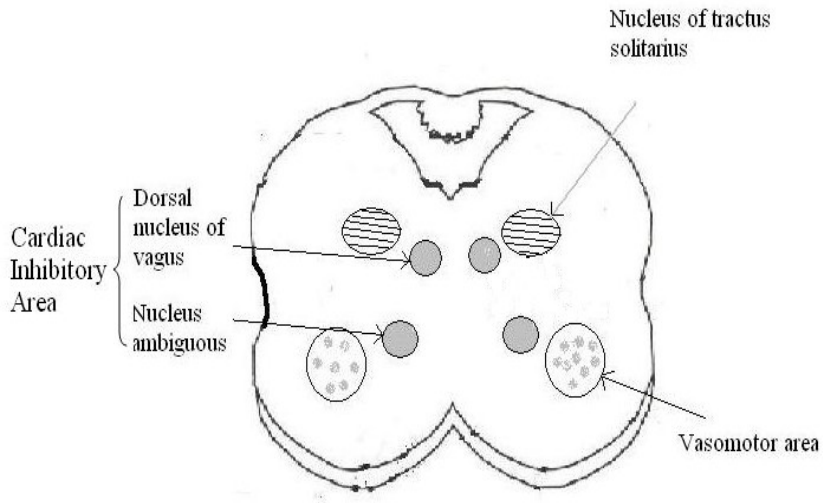

Figure (1): The medullary neurons that mediate sympathetic and parasympathetic discharge to blood vessels and heart. Nucleus tractus solitarius inhibits (-) vasomotor area and stimulates (+) cardiac inhibitory area.

Afferents to the medullary cardiovascular areas:

- These afferents change the activity of these areas (either increasing or decreasing their activity).
- The aim of these changes is to control the level of arterial blood pressure.

Afferents to the medullary cardiovascular centers that have a role in regulation of arterial blood pressure come from the following sites:

I. Arterial baroreceptors (high-pressure baroreceptors).
II. Cardio-pulmonary stretch receptors (low-pressure receptors).
III. Peripheral chemoreceptors from the carotid and aortic bodies.
IV. Central chemoreceptors.

In addition, the activity of medullary cardiovascular areas is affected by direct effects of hypoxia and hypercapnia.

I. Arterial baroreceptors (high-pressure baroreceptors).

These are mechanical stretch receptors. They act as "*pressure sensors*" that monitor the level of arterial blood pressure.

Site:

Arterial baroreceptors are located in:

- Carotid sinus (small dilation of the internal carotid just above the bifurcation of the common carotid artery).
- Aortic arch.

Innervation:

- Carotid sinus nerve (Hering's nerve) is a branch of the glossopharyngeal nerve (IX cranial nerve) that carries impulses from carotid sinus baroreceptors.
- Aortic nerve (branch of vagus, X cranial nerve).

- These are known as "***buffer nerves***" and they end in the *nucleus of tractus solitarius*.

Stimulation:

Arterial baroreceptors are stimulated by :

1. Arterial blood pressure: the threshold for baroreceptors stimulation is about 50 mm Hg. Higher pressure produces more discharge from baroreceptors. Maximal discharge rate occurs when pressure reaches about 160 mm Hg. Above that, no further increase in discharge rate occurs (figure 2).
2. Pulse pressure: higher pulse pressure causes more stimulation of baroreceptors than low pulse pressure. Baroreceptors are more sensitive to pulsatile pressure than to constant pressure. A decline in pulse pressure without any change in mean pressure decreases the rate of baroreceptor discharge.

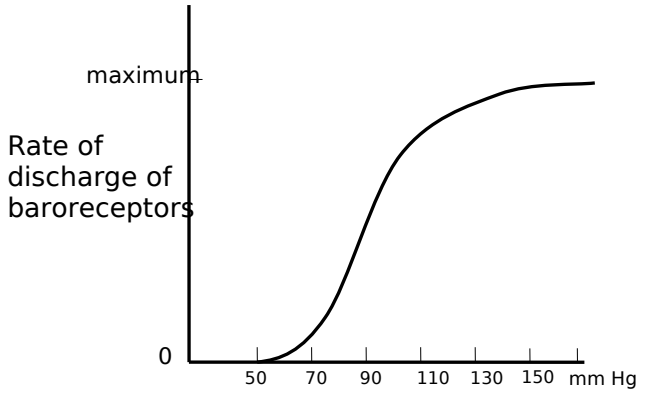

Figure (2): Rate of discharge of baroreceptors in response to different levels of arterial blood pressure

Function: Baroreceptor Reflex:

The function of baroreceptor reflex is to compensate for sudden changes in arterial blood pressure e.g. due to change in posture or hemorrhage.

1. At normal level of mean arterial blood pressure (90 mm Hg):
   Baroreceptors are stimulated and send low rate tonic impulses to relay on the nucleus of tractus solitarius (NTS) in medulla. Neurons of NTS send:
   a- Inhibitory signals to vasomotor area.
   b- Excitatory signals to cardiac inhibitory area (figure 3).
2. When arterial blood pressure increases:
   Tonic discharge from baroreceptors to NTS is increased (figure 3). This will lead to:
   a- More inhibition of vasomotor area. This will decrease sympathetic discharge to the heart (decreasing heart rate, stroke volume and cardiac output) and blood vessels (vasodilatation) leading to decrease of arterial blood pressure back to normal.
   However, if the elevation of arterial blood pressure is sustained for long time, the sensitivity of baroreceptor reflex decreases. This means that the reflex will function to maintain arterial blood pressure at this higher level. This is

referred to as "***resetting of baroreceptor reflex***". That is why baroreceptor reflex plays insignificant role in the long-term regulation of arterial blood pressure (e.g. defense against hypertension).

The "*set point*" of the baroreceptors reflex determines the level around which arterial pressure is regulated under different conditions. For example, during dynamic exercise arterial pressure is increased by approximately 15-20% and maintained at this level by changing the set point of the baroreceptors reflex. Resetting of the baroreceptors reflex is principally a centrally-mediated process that occurs within the neuronal connections of the receptors inside the central nervous system.

b- More excitation of cardiac inhibitory area. The vagal tone to the heart is increased leading to decreased heart rate and cardiac output. Arterial blood pressure will decrease back to normal.

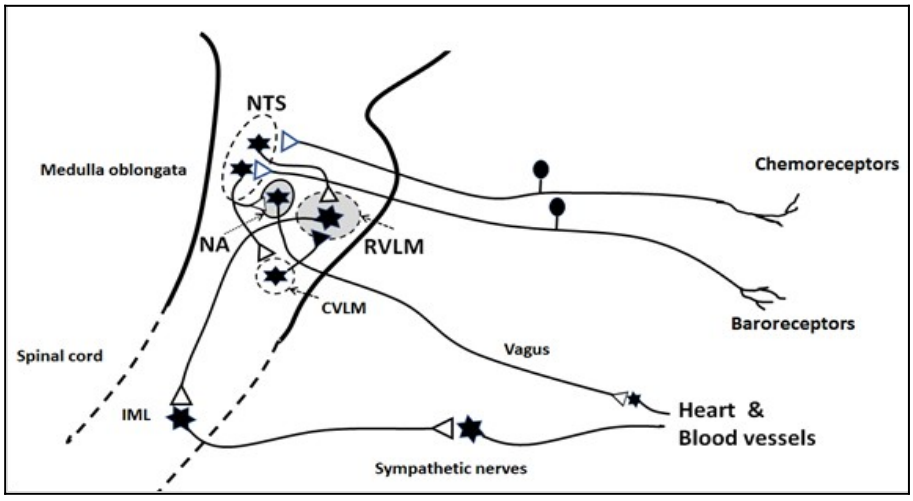

Figure (3): connections of NTS and medullary cardiovascular centers.

3. When arterial blood pressure decreases:

   Tonic discharge from baroreceptors to NTS is decreased. This will lead to:

   a- Less inhibition of vasomotor area. This will increase sympathetic supply to the heart (increasing heart rate, stroke volume and cardiac output) and blood vessels (vasoconstriction) leading to elevation of arterial blood pressure back to normal.

   b- Less excitation of cardiac inhibitory area. The vagal tone to the heart is decreased leading to increased heart rate and cardiac output.

II. Peripheral chemoreceptors:

Site:

- Peripheral chemoreceptors are located in carotid and aortic bodies.

Innervation:

- Chemoreceptors discharge is carried from carotid bodies by carotid sinus nerve (branch of the IX cranial nerve), and by the vagus nerve from aortic bodies.

Stimulation:

- These chemoreceptors are primarily concerned with respiratory regulation. They are more sensitive to low PO_2 than high PCO_2 or low pH (high H^+ concentration).

- However, if arterial blood pressure is markedly decreased to 40-60 mm Hg, blood flow in carotid and aortic bodies decreases. This leads to hypoxia and stimulation of the peripheral chemoreceptors.

Response:

- The response to peripheral chemoreceptor stimulation is to increase sympathetic discharge leading to tachycardia and vasoconstriction. This tends to elevate the low blood pressure.

III. Direct effect of changes in blood gases on arterial blood pressure:

Hypercapnia, and to a lesser extent hypoxia, can directly stimulate the vasomotor area leading to elevation of arterial blood pressure.

"***The Central nervous system (CNS) ischemic response***":

- Decreased arterial blood pressure (hypotension) leads to ischemia (decreased blood flow) of the medullary vasomotor area. Local PCO_2 rises. This will stimulate the vasomotor area leading to marked vasoconstriction and elevation of arterial blood pressure. The reflex is the most

powerful stimulus of the sympathetic nervous system. The reflex is excited only when arterial blood pressure drops below 50 mm Hg.

- **Cushing reflex:** This reflex is observed when intracranial pressure is increased. The patient has marked elevation of arterial blood pressure and bradycardia. ***Mechanism***: High intracranial pressure compresses cerebral blood vessels causing brain ischemia. The resulting hypercapnia and hypoxia produce a pressor response leading to elevation of arterial blood pressure. Cushing reflex is usually accompanied by bradycardia (through stimulation of baroreceptors.

Intermediate Time Course Mechanisms For the Regulation of Arterial Blood Pressure:

I. <u>Capillary Fluid Shift Mechanism:</u>

This mechanism operates to minimize the effect of changing blood volume on arterial blood pressure as follows:

a- If blood volume increases, blood pressure will also rise. However, increased blood volume leads also to rise of capillary hydrostatic pressure. This favors more filtration. Thus, blood volume decreases. Venous return and cardiac output decreases. The rise in arterial blood pressure is minimized.

b- On the other hand, if blood volume decreases the arterial pressure will decrease. Low blood volume decreases capillary hydrostatic pressure. This favors movement of fluid from interstitial space into capillaries. Blood volume increases. Venous return and cardiac output increase. The drop of arterial pressure is thus minimized.

II. <u>Renin-Angiotensin System (RAS) vasoconstriction</u>:

Decrease arterial blood pressure (ABP) and hypovolemia activate renin release from the juxtaglomerular apparatus in the kidneys. Renin acts on plasma angiotensinogen changing it to angiotensin I. Angiotensin converting enzyme (ACE) converts angiotensin I to angiotensin II. Activation of RAS and formation of angiotensin II takes about 20-30 minutes. Angiotensin II raises arterial blood pressure by direct vasoconstrictor action and by activation of the sympathetic discharge.

<u>III . Hormones ; Adrenaline , Nordrenaline ,Angiotensin II , Vasopressin .</u>

Long Term Regulation of Arterial Pressure:

Renal-Body Fluids Mechanism:

This mechanism operates over several days and is responsible for longer-term blood pressure control.

The volume of the extracellular fluid (ECF) is closely linked to arterial blood pressure.

Decreased ECF volume leads to decreased mean circulatory filling pressure, venous return, and cardiac output. This causes arterial blood pressure to decrease. Increased ECF volume has opposite effects, leading to elevation of arterial blood pressure.

The renal-body fluids mechanism controls the level of arterial blood pressure by adjusting the volume of ECF.

Volume of the ECF represents a balance between:

- Na^+ and water intake in diet.
- Na^+ and water excretion by kidneys.

Sodium intake depends largely on dietary habits and is not tightly controlled in humans.

Therefore, Na^+ balance and consequently ECF volume are maintained mainly by regulation of renal Na^+ excretion.

Renal-body fluid mechanism exerts its control on arterial blood pressure through the following mechanisms:

I. Renal pressure natriuresis:

a- When arterial pressure is elevated, renal excretion of Na^+ and water is increases (Figure 4). This is called “pressure natriuresis”. It results in decreasing the volume of ECF and lowering of arterial pressure. Pressure natriuresis continues until arterial pressure decreases to normal level.

b- When arterial pressure is low (e.g. after hemorrhage) renal sodium and water excretion decreases to minimize the decrease of arterial pressure.

- ***Pressure natriuresis mechanism is very powerful and is the basic and most important mechanism for long-term regulation of arterial blood pressure.***

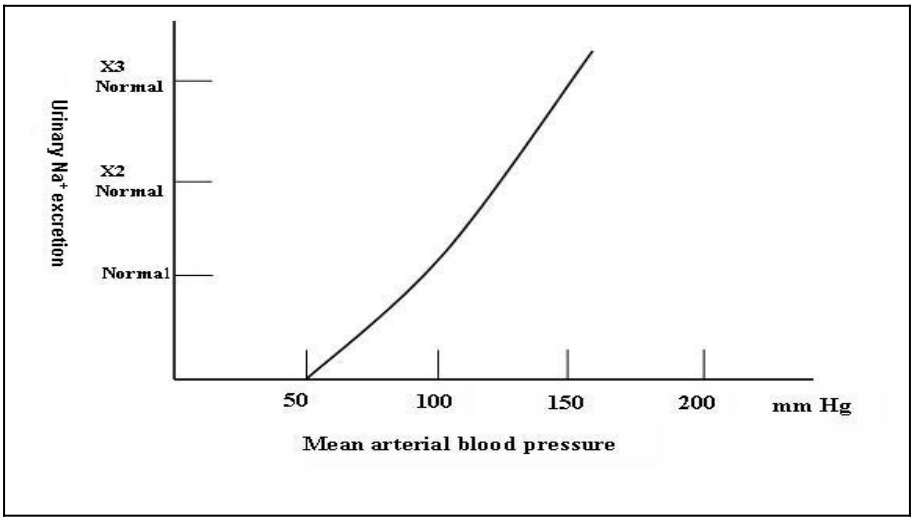

Figure (4): Renal pressure natriuresis. Sodium excretion increases when arterial blood pressure rises.

II. Renin-angiotensin-aldosterone system:

1- As explained before, decreased arterial pressure increase the secretion of renin. This results in formation of angiotensin II. Angiotensin II has an important role in long-term regulation of arterial blood pressure through:

a- It decreases Na^+ and water excretion by the kidneys.

b- It increases aldosterone secretion from the cortex of adrenal gland. Aldosterone is a powerful stimulator of Na^+

reabsorption by renal tubules. Na^+ and water excretion decreases. ECF volume increases and arterial pressure rises to normal.

2- On the other hand, rise of arterial pressure inhibits angiotensin II and aldosterone secretion leading to increased Na^+ and water excretion by the kidneys. ECF volume decreases resulting in decrease of blood pressure back to normal.

It should be noticed that inhibition of renin-angiotensin-aldosterone secretion enhances the activity of pressure natriuresis mechanism.

III. Atrial natriuretic peptides (ANP) secretion:

Increased ECF volume stretches atrial muscle fiber leading to secretion of ANP. ANP increases Na^+ excretion by the kidney and the volume of ECF decreases to normal. Blood pressure returns to normal.

It should be noticed that ANP enhances the activity of pressure natriuresis mechanism.

IV. Vasopressin secretion:

Decreased ECF volume results in decreased discharge of atrial low-pressure receptors. This leads to increased secretion of vasopressin which decreases water excretion by the kidneys (water retention). Vasopressin thus minimizes water excretion to prevent further decrease in ECF volume and arterial pressure.

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